Can the Wells-Riley model universally assess airborne pathogen infection risk?

Benjamin Jones*[1], Christopher Iddon[2], and Max Sherman[1]

1 University of Nottingham
Department of Architecture and Built Environment
Nottingham, UK
**Corresponding author:*
benjamin.jones@nottingham.ac.uk

2 University College London
Department of Civil,
Environmental and Geomatic Engineering
London, UK

ABSTRACT

Some airborne pathogens can infect susceptible people over long distances in buildings when they are transported in small respiratory particles suspended in the air. The pathogen concentration in air can be decreased using engineering controls, such as ventilation, filtration, or inactivation. To determine their effect, it is common to use the Wells-Riley model to estimate the probability that a susceptible person is infected and is a function of the dose of infectious pathogen received and a Poisson distribution. Wells proposed a hypothetical dose unit, known as the *quantum of infection*, which is a function of the pathogen emission rate and, in turn, a function of the number of infected people and their individual pathogen emission rates. The quanta generation rate can be determined from the epidemiological data for an outbreak case of a disease in a space where the proportion of a population of people infected with a disease who were initially free of it is known. The quanta generation rate is a temporally and activity varying parameter and so this approach only represents its value at the time the infections occurred and for that space. It is also unique for every disease and disease variant, and the emission rate varies in different spaces because the probability of the presence of infected people also varies. It is unknown at the start of a pandemic, and again later when the pathogen mutation period is greater than the time taken to determine uncertainty in its value. These factors make uncertainty in its value significant and it may vary by several orders of magnitude. A Monte Carlo analysis is used to show that uncertainty in the quanta emission rate for SARS-CoV-2 varies over around 8 orders of magnitude. There is a general paucity of data of sufficient quality to reduce uncertainty in emission rates. This means that there is little confidence in the data located in the tails. The problem with this is demonstrated by applying the emission rates to the WR model to estimate that, for an 8 hour exposure in a 50 person office with an outdoor airflow rate of 10 $l\,s^{-1}$ per person, the probability of infection of each occupant from long range transmission is <1% for 95% of events. It is the data in the right-tail of the emission rate distribution that leads to an appreciable probability of infection. These are just a few of several factors that make a probability of infection estimated by the Wells-Riley model unusable as a metric.

KEYWORDS

Quanta, infection risk, ventilation, far-field

1 INTRODUCTION

Some airborne pathogens can infect susceptible people over long distances in buildings when they contained in are transported in small respiratory particles with a range of sizes, some of which can remain airborne for long periods. When designing or controlling an indoor space that may contain infectious people, many of the factors of concern are fixed either by the health problem or by administrative requirements. However, the pathogens concentration in air can be decreased using various engineering controls, such as ventilation, filtration, or inactivation. To determine their effect, it is common to use the Wells-Riley (WR) model to estimate the probability that a susceptible person is infected and is a function of the dose of infectious pathogen received and a Poisson distribution. Wells proposed a hypothetical dose unit, known as the *quantum of infection*, defined as the number of infectious airborne pathogen required to infect 63% of susceptible people. Quanta comprises physical, biological, and statistical properties. It is a function of the pathogen emission rate and, in turn, a function of the number

of infected people and their individual pathogen emission rates. The quanta generation rate can be determined from the epidemiological data for an outbreak case of a disease in a space where the proportion of a population of people infected with a disease who were initially free of it is known. The quanta generation rate is a temporally and activity varying parameter and so this approach only represents its value at the time the infections occurred and for that space. It is also unique for every disease and disease variant, and the emission rate varies in different spaces because the probability of the presence of infected people also varies. It is unknown at the start of a pandemic, and again later when the pathogen mutation period is greater than the time taken to determine uncertainty in its value. These factors make uncertainty in its value significant and it may vary by several orders of magnitude. This could make a probability of infection estimated by the WR model unusable as a metric. Therefore, the aim of this paper is to quantify the uncertainty in both quanta emission rates and the probability of infection for SARS-CoV-2 to assess the ability of the WR model to universally assess airborne pathogen infection risk.

2 QUANTA DEFINED

The WR model describes the probability of becoming infected as a function of the dose of infectious agent received and a Poisson distribution (Riley 1978).

$$P(I) = 1 - e^{n} \tag{1}$$

Here, $P(I)$ is the probability of becoming infected and n is the quanta of infectious agent the individual is exposed to. This equation is the defining relationship and can be used to make a reasonable estimate of what its value must be. The WR model assumes that quanta and the infection probability is proportional to the dose of infectious agent received. The dose of many other infectious agents is given by the amount inhaled. Then the probability of infection can be expressed as

$$n = \int QC(t)\, dt \tag{2}$$

where t (h) is the time variable, C (quanta per m^3) is the concentration of infectious material the individual is exposed to, Q ($m^3\ h^{-1}$) is the breathing rate of an exposed person. This equation assumes that all uninfected people are equally susceptible, and they are not wearing personal protective equipment, such as a mask.

To follow an individual, the integration over time reflects what they are doing, and the quantities within it vary. To evaluate what is happening for a specific activity, Equation (2) can be simplified further by representing the quantities within the integral with their averages. Then,

$$n = \bar{Q}\bar{C}D \tag{3}$$

where D (h) is the duration of the activity and the overbars of the quantities indicate the time average over that duration.

To find the average concentration, we follow the WR model and make the assumption that the infectious material is conserved, but with a first order loss term ϕ (h^{-1}), and the only source of infectious material is infected people. In this case, the time evolution of the concentration is determined by a first order linear differential equation with the time variable assumed.

$$\frac{dC}{dt} + \phi C = \frac{1}{V}\sum_{i=1}^{j} q_i \tag{4}$$

Here, V (m^3) is the volume of the space, and q_i (quanta h^{-1}) is the emission rate of each of j people in the space, which is zero for those uninfected. The loss term is the sum of the outside

air change rate ψ (h^{-1}), the biological decay rate of the pathogen λ (h^{-1}), and the surface deposition rate of respiratory particles γ (h^{-1}).

$$\phi = \psi + \lambda + \gamma \tag{5}$$

The viral load of an infector evolves through the course of their infection with a particular disease; see Cevik *et al.* (2020) for SARS-CoV-2, similarly the emission rate is also seen to evolve see Zhou *et al.* (2023). It should be noted that the WR model assumes that there is an equivalent number of infected people, and that an infected person emits at a fixed rate. The emission rate term might also be reduced by any source removal mechanism, such as local capture or filtration immediately adjacent to an infected person, but it is not included here.

The average concentration can be calculated from the standard solution to a first order differential equation, but we can make the simplifying assumption that it can be treated as a steady state, when it is possible to remove the overbars so that

$$n = \frac{QD\sum_{i=1}^{j} q_i}{\phi V} \tag{6}$$

The quanta emission rate, q_i (quanta h^{-1}), for a single infected person can be calculated as a function of the emission rate of viable virions, G (virions h^{-1}), the fraction of virions absorbed by the respiratory tract of a susceptible person after they enter, k, and the dose constant, K, the reciprocal of the probability that a single virion initiates an infection.

$$q \equiv kG/K \tag{7}$$

If the breathing rate of a single infected person is the same as that for susceptible people, G is determined by adapting Buonanno *et al.* (2020) to be

$$G \equiv QV^*_{drop}Lv \tag{8}$$

Here, L (RNA copies m^{-3}) is the load of viral genomic material in the respiratory fluid, much of which is genomic material and not viable virus, and so v (virions per RNA copy) is the viable fraction. V^*_{drop} is the total volume (m^3) of expelled airborne respiratory particles (respiratory fluid) in 1 m^3 of exhaled air and is a function of the number of all respiratory particles per unit volume of exhaled air, C_{drop} (# m^{-3}), and the mean diameter of the exhaled respiratory particles, $\bar{d}$ (m). An assumption is made about the hydrated volumes of the respiratory particles measured in experiments used to derive $\bar{d}$ (see Morawska *et al.* 2009) and so an evaporation term, E, is used to account for the hydrated volume of V^*_{drop}.

$$V^*_{drop} \equiv \frac{\pi}{6}\left(\bar{d}E\right)^3 C_{drop} \tag{9}$$

3 UNCERTAINTY IN THE QUANTA EMISSION RATE

The derivation in Section 2 treats all parameters as being known precisely, although few of them are. Some, like the duration (D), can be assumed to be known because they are set as part of the design process. Others, such as the quanta emission rate (q), are known poorly. This type of parameter is best described by a distribution. Furthermore, it is preferable to predict a probability distribution for $P(I)$ rather than a simple maximum likelihood estimate. It is also the only meaningful way to understand $P(I)$ when parameter values are unknown at a moment in time, or when it is desirable to understand the uncertainty in the general risk of being in a particular space. Given that the model is the product of many assumedly uncorrelated terms, the distribution of its predictions is expected to be approximately log-normal whose variance is best described by a geometric standard deviation.

Each term in Equations 5-9 is considered separately and appropriate values and distributions are given in Table 1. The rate of aerosol emission and their origin in the respiratory tract is a function of respiratory activity, such as breathing, talking and vocalisation (singing an "*aaah*") (Morawska *et al.* 2009). Aerosol diameters range from <1 μm to >100 μm, and their size distribution is dependent upon the expiratory activity, usually following a log-normal distribution (Morawska *et al.* 2009). Larger respiratory particles fall ballistically under gravity in still air. Respiratory particles with an evaporated diameter of <10 μm can remain airborne for several hours. The mean aerosol diameter ($\bar{d}$) is derived experimentally for people breathing for 75% of the time and talking for 25% (Morawska *et al.* 2009), and their distribution is assumed to be log-normal with a mean value of 1.84×10^{-6} m and an arbitrary standard deviation of 10% of the mean. Respiratory particles evaporate reducing their diameter, mass, and terminal velocity. Therefore, the diameter of the respiratory particle is likely to be greater when emitted (hydrated) than when measured and this is accounted for by the evaporation factor (E) that has limits of 2 and 5, but Nicas *et al.* (2005) advise that this is likely to be closer to 2 than 5 and so a beta distribution is used with α=2 and β=5.

The number of all respiratory particles per unit volume of exhaled air (C_{drop}) is also given by Morawska *et al.* (2009) whose data contains concentrations of evaporated respiratory particles with a diameter of <5 μm measured during breathing, talking and vocalisation. We assume 25% talking and 75% breathing and that C_{drop} is log-normally distributed with a mean of 9.8×10^{4} respiratory particles m^{-3} and has an arbitrary standard deviation of 10% of the mean.

The viral load (L) of an infected person increases with time from the moment of infection peaking just before, or at, the onset of symptoms and decreases thereafter, normally ceasing within a week of the onset of symptoms (Cevik 2020, Cevik 2021). The magnitude of L also varies widely between people at any stage of the infection, which increases uncertainty in it (Killingley *et al.* 2022). Iddon *et al.* (2022) show the distribution of L within an infected population is unknown. Therefore, we arbitrarily use the data of Chen *et al.* (2021b) who predict that $\log_{10}$ values of L taken from NP swabs of individuals 2 days from symptom onset are

Table 1: Uncertainty in input parameters

	Variable	Values	Source
Quanta emission rate	Breathing rate, Q (m^3 h^{-1})	LN(0.56, 0.056)	(Adams 1993)
	Respiratory activity, *breathing:talking* (%)	75:23	(Morawska *et al.*,2009; Iddon *et al.* 2022)
	Aerosol concentration in exhaled air, C_{drop} (respiratory particles m^{-3})	LN(1.54×10^{5},1.54×10^{4})	(Morawska *et al.*,2009; Iddon *et al.* 2022)
	Mean aerosol diameter, $\bar{d}$ (m)	LN(1.91×10^{-6},1.91×10^{-7})	(Morawska *et al.*,2009)
	Aerosol evaporation factor, E	B(2.0,5.0) [2.0,5.0]	(Nicas *et al.* 2005)
	Viral load, L ($\log_{10}$ RNA copies ml^{-1})	N(7.0,1.4)	(Chen *et al.* 2021)
	Viable fraction, v	B(2.0,5.0) [10^{-4},10^{-2}]	(Killingley *et al.* 2022)
	Respiratory tract absorption fraction, k	U(0.43,0.65)	(Darquenne 2012)
	Dose constant, K	U(5,15)	(Killingley *et al.* 2022)
Scenario	Number of infected people, j	1	
	Number of occupants	50	
	Space volume, V (m^3)	1350	
	Exposure duration, D (h)	8	
	Outside airflow rate (l s^{-1} per person)	10	
	Outside air change rate, ψ (h^{-1})	1.33	
	Biological decay rate, λ (h^{-1})	LN(0.63,0.43)	(Van Doremalen *et al.* 2020)
	Surface deposition rate, γ (h^{-1})	U(0.42,0.61)	(Thatcher *et al.* 2002)

N(μ,σ), normal(mean, standard deviation); LN(μ,σ), log-normal; U(max,min), uniform; B(α,β) [min, max], beta.

Note that L needs to be converted into RNA copies per m^3 by multiplying by 10^6.

normally distributed with a mean of 7 and a standard deviation of 1.4 $\log_{10}$ RNA copies ml^{-1}. The reported values of viral load are per ml of the sample medium rather than the respiratory fluid, but it is a good surrogate for the range of viral loads.

The viable fraction (v) is not well understood, but it changes as the disease progresses, and there is heterogeneity between patients. Killingley *et al.* (2022) show that a conservative estimate of v is between 1: 10^{-2} and 1: 10^{-3} viable virions to RNA copies, but it could be as high as 1:10^{6}. We assume that v is closer to 10^{-2} using a beta distribution with a lower limit of 10^{-4} and arbitrary values of α=2 and β=5 to give a mean of 2.9×10^{-2}.

The respiratory tract absorption fraction (k) is a function of aerosol diameter and the breath volume. For the mean $\bar{d}$ described earlier, Darquenne *et al.* (2012) estimates k has a range of 0.4—-0.65 and, in the absence of knowledge, we assume that all values are equally probable between these limits.

There is no direct measured value of a dose constant (K) for the SARS-CoV-2 virus. A SARS-CoV-2 human challenge infected 53% of participants with a nasally applied solution of 10 $TCID_{50}$ (50% Tissue Culture Infectious Dose)[1] of viable SARS-CoV-2 virus (Killingley *et al.* 2022, Zhou *et al.* 2023). A crude estimate is that 1 $TCID_{50}$ is equivalent to 0.7 PFU indicating that the dose was 7 PFU and for the proportion infected would suggest K is approximately 9.3, for this method of application. Here, we assume that 1 PFU is equivalent to a single viable virion, although it is possible that greater than 1 viable virion is required to lead to a plaque due to probabilities of binding to the correct receptor, cell fusion and giving rise to a successful infection. Therefore, we assume K is equally probable between 5 and 15 as a reasonable assumption based on this data point.

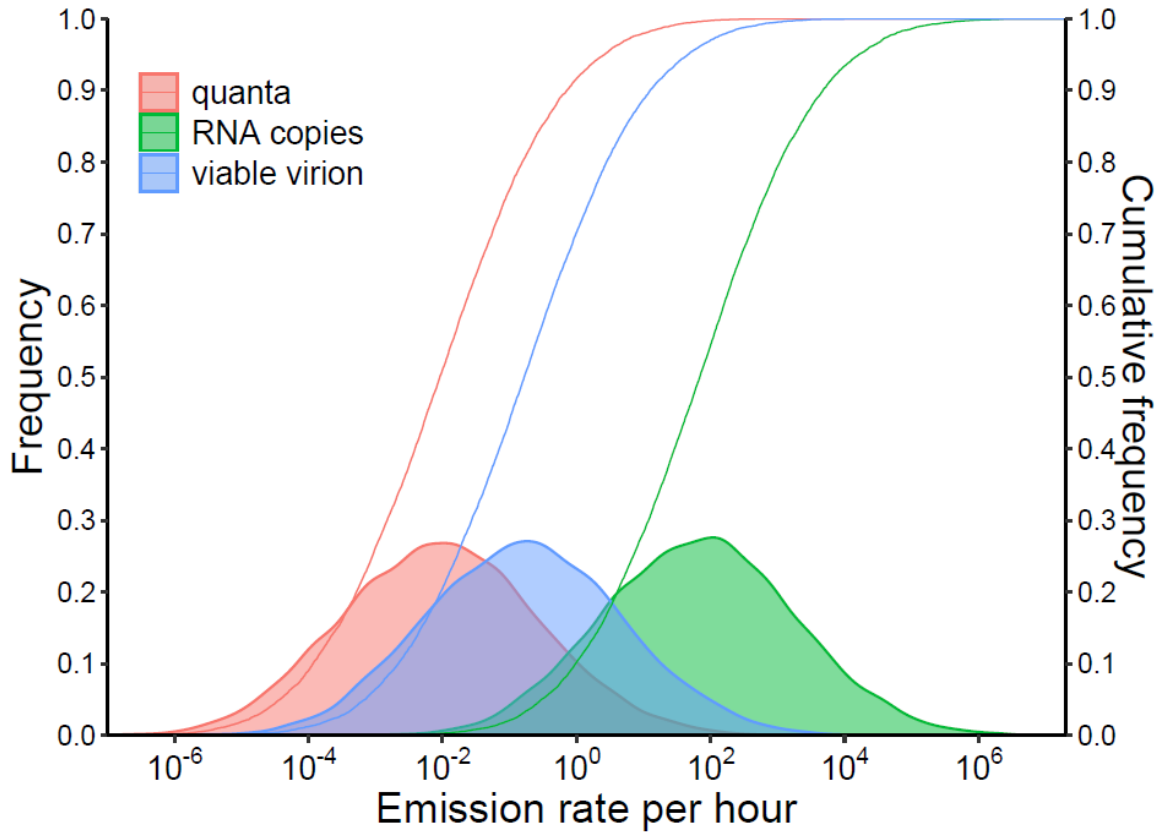

Figure 1: Emission rates of quanta, RNA copies, and viable virions (all per hour).

[1] One $TCID_{50}$ quantifies the amount of virus required to produce a cytopathic effect in 50% of inoculated tissue culture cells.

Table 2: Uncertainty in calculated parameters

	GM	GSD
Expelled volume ratio of respiratory particles to air, V^*_{drop}	5.9×10^{-12}	1.8
Viable virion emission rate G (virions h^{-1})	8.0×10^{-2}	28
Quanta emission rate q (quanta h^{-1})	3.5×10^{-3}	28
Removal rate, ϕ (h^{-1})	2.4	1.2
Equivalent ventilation rate (l s^{-1} per person)	18	1.2
Probability of infection, $P(I)$	4.7×10^{-6}	29

GM: Geometric Mean; GSD: Geometric Standard Deviation

It is possible to estimate the uncertainty in the quanta emission rate (q) for a single infected person undertaking some activity that is independent of the space they occupy. Personal factors, such as L, vary relative to the population mean but average out when considered over the entire population distribution. Scenario specific factors, such as Q, C_{drop}, and $\bar{d}$, do not average out when considered for a population and are, therefore, unknowable. None of these parameters depend on space geometry or outside air delivery rates. Accordingly, when the quanta emission rate inputs identified in Table 1 are applied to Equations 5-9 using a Monte Carlo approach (3 consecutive sets of 10^5 samples produce identical geometric means for q and $P(I)$ when rounded to 3 significant figures), the geometric means (GM) and standard deviations (GSD) of V^*_{drop}, G, and q are calculated and given in Table 2. A probability density function (PDF) and cumulative distribution function (CDF) of q is given in Figure 1.

Figure 1 shows that q varies over around 8 orders of magnitude. The 95% confidence interval for q is between 1.4×10^{-5} and 7.0 quanta h^{-1}, which shows that the uncertainty in its value is around 5 orders of magnitude most of the time. The upper interval seems low when compared to those reported for SARS-CoV-2 superspreading events; for example, the 970±390 quanta h^{-1} reported by Miller *et al.* (2020) and the 130 quanta h^{-1} (inter-quartile range of 97-155 quanta h^{-1}) reported by Vernez *et al.* (2021). Figure 1 shows that this range of values is possible for a single index case, but only for around 0.1% of the time. There is likely to be a difference in the breathing rate and the respiratory activity of the occupants reported by Miller *et al.* and Vernez *et al.* and those described here, which effect the mean aerosol diameter and the aerosol concentration in exhaled air. Future work will compare q against new empirical analyses of emission rates, such as human challenge studies.

4 UNCERTAINTY IN PROBABILITY OF INFECTION

The probability of infection, $P(I)$, in a common space type can be demonstrated using an example scenario. We consider an office space with a floor to ceiling height of 2.7m and an occupancy density of $10m^2$ per person, which contains 50 unmasked occupants who are present for 8 hours. It is mechanically ventilated with an outside delivery rate of 10 l/s per person without filtration. A single infected person is assumed present for the duration. Equation 1 is used to estimate $P(I)$. All inputs are given in Table 1 and outputs are in Table 2. The probability of infection of each occupant from a single infected person from long range transmission is <1% for 95% of the time. There are problems with this, such as the assumption of a single infected person, and in the significant uncertainty in the tails of the distribution of q, which is discussion in Section 5. It does show, however, the need to understand q better.

5 DISCUSSION

The example highlights problems with inferring quanta empirically for any airborne pathogen and applying it in the same location for the same scenario. First, it isn't always possible to know the number of infected people. The probability of any number of infected people increases with the community infection rate and the number of occupants Iddon *et al.* (2022). For low

community infection rates, the most likely number of infected people in common spaces is zero. Furthermore, the viral load varies by time and person, depending on the stage of a disease, inter-person viral dynamics and the immune response. Secondly, if the space remains unchanged but the scenario differs, then the duration and respiratory activity may vary, and consequently so do the distributions representing the breathing rate, the aerosol diameter, and the concentration of respiratory particles in exhaled air. Thirdly, for a scenario in a new space, the volume and occupancy density may change in addition to all other parameters, and consequently so does the *equivalent* ventilation rate (ϕ). Finally, by far the biggest problem that the data analysis shows, is a general paucity of data of sufficient quality to reduce uncertainty in q and $P(I)$. At the start of a pandemic the viral load, L, and hence q, are always unknown. Once a population begins to acquire immunity, the proportion of occupants susceptible to infection reduces. When infections do not confer sterilising immunity, the proportion of susceptible people is then dependent on immunity waning and the ability of a pathogen to mutate to evade immunity. The consequence of the low quality of data is that we do not know the true underlying distributions of either the inputs or the outputs and so we have little confidence in the magnitudes of the tails where there is very little supporting data. Accordingly, there is much uncertainty in the tails, and in the confidence intervals given here. They should, therefore, be considered very approximate. We report both the GM and the GSD because the distributions of the predictions are thought to be log-normal (see Section 2) and indicate where the majority of data lies and where there is more confidence in it. It is important to note that other values and distributions could be used, but these outcomes will still be true. This also applies to distributions for other pathogens, particularly those that are less well studied than SARS-CoV-2.

There are also more general and fundamental issues with inferring quanta empirically. The WR model estimates the infection risk from long range transmission, but it impossible to disaggregate it from other exposure pathways. Furthermore, quanta emission rates are derived from observational studies of high secondary attack rates of transmission resulting in the calculation of high magnitudes, which are then extrapolated to most other cases where its magnitudes are low, which introduces significant bias. Many models of infection risk use specific values of q determined from outbreaks. Figure 1 shows that q is a continuum that spans 8 orders of magnitude and so it needs to be used to estimate $P(I)$, rather than using specific values of q, if the estimate is to have any context and meaning. Even then, a distribution of $P(I)$ should only be used to show where most predictions lie.

Together, these factors make an absolute value of the personal risk of long-range airborne infection probability, $P(I)$, unusable as a metric for SARS-CoV-2 or any other airborne pathogen. It can, however, be used to give an appreciation of the magnitude of absolute risks, which we estimate to be low most of the time. When accounting for the likelihood of the presence of an infected person, the magnitude of absolute risk reduces even further; see Iddon *et al.* (2022).

6 CONCLUSIONS

The quanta emission rate of an infected person is a continuum that varies over time and between people. We find that the uncertainty in the magnitude of quanta emission rate for SARS-CoV-2 for a single infected person varies by over around 8 orders of magnitude. Unfortunately, there is a general paucity of data of sufficient quality to reduce uncertainty in emission rates. This means that there is little confidence in the data located in the tails. The problem with this is demonstrated by applying the emission rate continuum to the WR model to estimate that, for an 8 hour exposure in a 50 person office with an outdoor airflow rate of 10 l s^{-1} per person, the probability of infection of each occupant from long range transmission is <1% for 95% of events. It is the data in the right-tail of the distribution of the quanta emission rate, where there is little confidence in their magnitudes, which leads to an appreciable probability of infection.

Therefore, it is impossible to say, with any certainty, the fraction of events that will lead to some probability of infection.

These factors make an absolute value of the personal risk of long-range airborne infection probability unusable as a metric for SARS-CoV-2 or any other airborne pathogen. It can be used to give an appreciation of the magnitude of absolute risks, which we estimate to be low most of the time.

7 ACKNOWLEDGEMENTS

The authors acknowledge the Engineering and Physical Sciences Research Council (EP/W002779/1) who financially supported Jones and Iddon.

8 REFERENCES

W. Adams, Measurement of breathing rate and volume in routinely performed daily activities, Final report, contract no. a033-205., California Air Resources Board, Sacramento (1996).

G Buonanno, L. Stabile, L. Morawska, Estimation of airborne viral emission: Quanta emission rate of SARS-CoV-2 for infection risk assessment, Environment International 141 (May) (2020) 105794.

M. Cevik, K. Kuppalli, J. Kindrachuk, M. Peiris, Virology, transmission, and pathogenesis of SARS-CoV-2, The BMJ 371 (2020) 1–6.

M. Cevik, M. Tate, O. Lloyd, A. E. Maraolo, J. Schafers, A. Ho, SARS-CoV-2, SARS-CoV, and MERS-CoV viral load dynamics, duration of viral shedding, and infectiousness: a systematic review and metaanalysis, The Lancet Microbe 2 (1) (2021)

P. Z. Chen, N. Bobrovitz, Z. Premji, M. Koopmans, D. N. Fisman, F. X. Gu, SARS-CoV-2 shedding dynamics across the respiratory tract, sex, and disease severity for adult and pediatric covid-19, eLife 10 (2021) e70458.

C. Darquenne, Aerosol deposition in health and disease, Journal of Aerosol Medicine and Pulmonary Drug Delivery 25 (3) (2012) 140–147.

C. Iddon, B. Jones, P. Sharpe, M. Cevik, S. Fitzgerald, A population framework for predicting the proportion of people infected by the far-field airborne transmission of SARS-CoV-2 indoors. Building and Environment. 2022;221:109309.

B. Killingley *et al.* Safety, tolerability and viral kinetics during SATS-CoV-2 human challenge in young adults, Nature Medicine 28 (5) (2022) 1031–1041.

S. L. Miller *et al.*, Transmission of SARS-CoV-2 by inhalation of respiratory aerosol in the Skaagit Valley Chorale superspreading event, Indoor Air 31 (2021) 314-323.

L. Morawska, *et al.*, Size distribution and sites of origin of droplets expelled from the human respiratory tract during expiratory activities, Journal of Aerosol Science 40 (3) (2009) 256–269.

M. Nicas, W. W. Nazaroff, A. Hubbard, Toward understanding the risk of secondary airborne infection: Emission of respirable pathogens, Journal of Occupational and Environmental Hygiene 2 (3) (2005) 143–154.

E. C. Riley, G. Murphy, R. L. Riley, Airborne spread of measles in a suburban elementary school, American Journal of Epidemiology 107 (1978) 421–432.

T. L. Thatcher, A. C. Lai, R. Moreno-Jackson, R. G. Sextro, W. W. Nazaroff, Effects of room furnishings and air speed on particle deposition rates indoors, Atmospheric Environment 36 (11) (2002) 1811–1819.

N. Van Doremalen *et al.*, Aerosol and surface stability of SARS-CoV-2 as compared with SARS-CoV-1, New England Journal of Medicine 382 (16) (2020) 1564–1567.

D. Vernez, S. Schwarz, J. J. Sauvain, C. Petignat, G. Suarez, Probable aerosol transmission of SARS-CoV-2 in a poorly ventilated courtroom, Indoor Air 31 (6) (2021) 1776–1785.

J. Zhou *et al.*, Viral emissions into the air and environment after sars-cov-2 human challenge: a phase 1, open label, first-in-human study., The Lancet. Microbe 6 (2023).